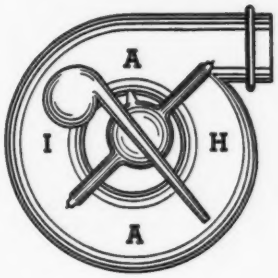
AMERICAN

INDUSTRIAL HYGIENE

ASSOCIATION

QUARTERLY



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This Issue

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Forty Years in the Poisonous Trades

ALICE HAMILTON, M.D.

I AM afraid that the title of my paper may give the impression that I mean to indulge in the vice of old age and waste my time on tales of the far-distant past. I will not do that but I cannot resist the temptation to give you of the later generation something of a picture of what industrial medicine was 40 years ago. For only if you look at it can you appreciate the remarkable record your country has made in that really brief period, a record starting in ignorance almost complete, and willfully so, to a position of what we may almost call world leadership, the countries which for so long led us being now unable to keep pace because of war destruction and poverty.

When I entered this field in 1910, after two years study of all the available English and German material, industrial medicine did not exist in the U.S.A. and industrial surgery was rather discredited. I think you could have counted the published articles on industrial poisoning on the fingers of one hand. There were of course men in industry, foremen especially, who had a lot of practical experience, and a few doctors really studied their cases, but the prevailing attitude was ostrich-like. American factories were so much better than European, American workmen so much better housed and fed than European, that the industrial diseases described by foreign authorities simply did not exist over here. I remember the shocked surprise I felt when I visited the model town of Essen, after I had seen South Bethlehem.

The most difficult obstacle I found was the universally held doctrine that the only way to prevent industrial poisoning was to keep the skin clean, to scrub the hands and especially the fingernails. I have been reading over some of the papers I wrote before the First World War and in all of them I plead with physicians and employers to give up the clean fingernail doctrine and face the fact that a lead worker eats only three times a day and even then he does not wash his hands in his soup or coffee, but he breathes 16 times a minute and if there is lead in the air, he will get it no matter how often he scrubs his nails. But it was uphill work, for I was trying to upset a cherished myth. I remember the head surgeon of a great Colorado smelting company saying in a public meeting: "It is not the lead a man absorbs during his work that poisons him but what he carries home on his skin." I remember the physician in charge of a Utah smelter telling me he always instructed the men—blast furnace tenders, flue and baghouse cleaners—to scrub their nails carefully.

At that time we believed that the lead which was inhaled reached the stomach by being mixed with the saliva and swallowed and the only reason why men working in fumes and dust were intoxicated so much more rapidly and severely than those handling solid lead (a fact noted by Tanquerel des Planches almost a century ago) was that the quantity entering the body was greater. K. B. Lehmann, then one of our greatest authorities, thought he had proved this by a few questionable human experiments. It was not until J. C. Aub made his studies in the twenties that we understood the difference between absorption

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Presented as the Cummings Memorial Lecture at the Ninth Annual Meeting of the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION, April 1, 1948, Boston, Massachusetts.

through the gastro-intestinal tract, with its many natural defenses, and that through the almost completely defenseless respiratory tract. These studies really revolutionized the attitude of physicians and of practical men towards the protection of lead workers. For many years I believed that the body cleanliness story was a thing of the past and then not long ago I picked up a publication of one of our governmental industrial hygiene outfits, and read: "In the prevention of benzol poisoning the most important element is bodily cleanliness." Sometimes it seems as if a lie crushed to earth will rise again. Imagine a man who has to breathe benzol fumes protecting himself by bathing.

The earliest method of protection against severe exposure to an industrial poison was to shorten the time of exposure and the first such measure of which we know was introduced in 1556 in the Austrian mercury mines of Idria. The working day was shortened by law to six hours. In Spain, in the great quicksilver mines of Almaden as late as 1921 30 hours constituted a month's work and in England in 1899 strippers of the white bed in white lead works were forbidden to do that work on more than two successive days. Practically the only method of prevention I found before the First World War in lead smelting and in the production of white lead and in roasting oxides was to rush the dangerous jobs and then discharge the men. It was always a matter for the foremen to decide, the management knew nothing about it except that there was an enormous labor turnover. But in those days that was a matter for pride. Foremen would boast of having to bring in from 30 to 50% new men every pay day. In that way, they thought, not many would get severely leaded. Of course there were always plenty of newly arrived immigrants to fill the ranks.

Even now the shortening of exposure may be adopted as the only practical way of avoiding serious trouble. There are emergencies, such as accidental spillage, or breakage, or unusual repair work, which have to be dealt with at once. It may be that the only possible way to manage is to let no individual work in the poisoned air for more than a few minutes.

Dr. Alice Hamilton, Cummings Lecturer for the Ninth Annual Meeting of the American Industrial Hygiene Association, is the world's foremost woman industrial hygienist. Whenever workers have been subjected to poisonous materials in their trades, Dr. Hamilton has brought the facts to light by visiting the plants and talking with the affected workers. She has continually fought for control of industrial health hazards through legislation, by convincing management to take action, and through education. Her investigations have included the lead industries, viscose rayon production, potteries, steel mills, mining.

The first woman on the faculty of Harvard Medical School, the only woman to serve on the Health Committee of the League of Nations and to receive the American Public Health Association's Lasker Award for combatting causes of disease and death, Dr. Hamilton is author of several books on industrial toxicology. She is an active member of the Committee on Permissible Limits of Dusts, Gases, and Vapors of the American Standards Association. Her work over the past forty years has done much to demonstrate the necessity of vigorous, scientific control of poisonous materials incident to the occupation.

The Cummings Memorial Lecture is given annually by an outstanding personality in the field of industrial hygiene in commemoration of Donald E. Cummings, Past-President of the American Industrial Hygiene Association, killed in a plane crash en route to control an occupational disease situation jeopardizing production of a critical war material.

Two subjects that in the early days bothered us a great deal were the special danger to young people from industrial poisons and the special danger to women.

As to the first, the great susceptibility of youth is admitted and the Federal Childrens' Bureau has for some years examined the industries employing minors and secured *gentlemen's agreements* to rule out minors from possibly hazardous jobs. When it comes to the over-susceptibility of women, a theory which has prompted some quite foolish legislation, careful observation of large groups in the poisonous trades has not given proof of it. We still hold that plumbism is more harmful in a woman because it is a poison to the germ cell and although that is true too of the male germ cell, still in the woman there is the additional injury to the fetus by the lead in the mother's blood while she is carrying the child. This is probably true also of mercury. As to benzol we still believe that it is more harmful to menstruating girls than to young men, though we are not so strongly convinced now as we used to be. Certainly there is no ground for forbidding women to work in jobs that expose them to carbon monoxide or to silicosis. Statistics seem to point the other way.

Lead

IT IS in the industrial control of lead poisoning that we see, I think, the greatest advance over the situation that obtained in this country up to the First World War. Let me give briefly a few pictures of the chief lead industries as they were then, reminding you that whatever statistical information we had was based on personal surveys including interviews with physicians, labor leaders, social workers, druggists, and on hospital records, not on routine medical examination of the individuals in lead work.

The production of white lead and of the oxides was the most notorious of the lead trades and in every center it was possible to pick up reports of palsies, encephalopathies, deaths from severe intoxication. That industry has been revolutionized, the dusty processes controlled, the workers kept under medical supervision. In the old days, Philadelphia and Chicago had so many cases of plumbism from white lead and red lead plants that Cook County Hospital and old Blockley listed them under the name of the plant, not under lead poisoning—that was taken for granted.

Pottery glazing and decorating was another notorious source for we used white lead unchanged in the glaze and the colors and made no effort to control the dust. It was then that I came across my first cases in women, dippers' helpers and lithotransfer makers, and convinced myself that women are more susceptible than men to cerebral plumbism. Then I went to England and Germany and saw what could be done with good exhausts and with wetness everywhere. I saw how the English changed soluble white lead in the glaze by fritting it to insoluble disilicate. We had in 1922 a rate of 22 to 23% of plumbism among potters, the English had a rate of 0.9%. Now we frit our glazes and we practice good housekeeping in our potteries, so Ohio, New Jersey and West Virginia are no longer happy hunting grounds for the student of plumbism. An equally great reform has occurred in the making of sanitary ware. That used to seem to me the worst of the lead trades. The enamelers scattered a powder containing up to 20% soluble lead over red hot iron sinks and bathtubs, the men and all surfaces were dust-covered. In one Pennsylvania plant employing 130 enamelers, doctors and hospital records showed that in two years there had been 72 cases of severe plumbism, with five of palsy, two of encephalopathy and three deaths. Now we use a leadless enamel on clay bathtubs and sinks.

The rubber industry was once a source of severe forms of plumbism, because lead oxides and the basic carbonate were important compounds and men in the compounding rooms and on the mills were exposed to soluble lead in the form of very fine dust. Now lead has almost vanished from the rubber industry.

The old notorious lead trades, painting, printing, plumbing, which gave rise to the expressions "painters' or printers' colic and palsy," are no longer important in that respect. The newer coatings, enamels, lacquers, plastics, are lead-free, titanium dioxide displaces white lead to a great extent in paint, and interior decoration is done with leadless paint. Printing plants

are far better equipped and largely mechanized, plumbers hardly come in contact with lead except for rare jobs.

Yet lead still holds an important place in the list of industrial poisons for though it vanishes from an old trade, it may suddenly appear in a new one. Painting automobiles with many coats of lead paint gave way to lead-free lacquers, but when streamlining came in, the lead problem made a dramatic reappearance for the surfaces of the car must be absolutely smooth and this means that seams must be covered with lead solder and then sandpapered and smoothed. The scrapping of steel structures by the use of intense heat is an increasing source of danger. Recently Cotter described some 300 cases of "lead intoxication by inhalation" in men scrapping the elevated railroads in New York by using blow torches on the steel which had had many coats of red lead paint. Even more dangerous because it is often carried on in enclosed places and with extreme degrees of heat is welding, which is more and more replacing riveting. Tetraethyl lead is an ever-present danger, though scrupulously safeguarded now. And the trade which in all industrial countries yields the greatest number of cases of plumbism, the making of storage batteries, is not only still with us but increasing in importance. It is a very difficult problem for the industrial hygienist. I have never seen a plant which was entirely safe, though the English system of keeping everything dripping wet has much in its favor. Belknap of Milwaukee puts his mixers, pasters and finishers into positive pressure masks, but I believe that even this drastic system is not entirely foolproof. We are told that a new kind of lead-free storage battery, developed by the French, is being introduced in this country but since it uses cadmium, it is rather like leaping from the frying pan into the fire.

There are questions still unsettled with regard to lead poisoning in spite of the enormous amount of study which has been devoted to the subject: the possible relation between lead poisoning and peptic ulcer, between lead poisoning and multiple sclerosis, arteriosclerotic kidney, generalized arteriosclerosis. Here the English are doing the most significant work, for they need not depend on animal experiments, they can follow human lead workers over a long period of years, men and women who worked in the lead trades as much as 50 years ago before sanitary control was perfected, and who at the end show the effects of what the English believe to be slow, prolonged poisoning. The English law accepts as occupational in character chronic nephritis with or without general arteriosclerosis in an old lead worker.

Unfortunately for us, during the years when lead poisoning of a severe character was prevalent in this country, no real interest was taken in its study by physicians. It was not till hygienic reform was well under way and severe poisoning controlled that we began intensive research.

Mercury

THE old familiar poison, mercury, has held its place in industrial toxicology since Roman days when the quicksilver mines of Spain were exploited, but the industry most notorious for its disastrous effects has always been the making of felt hats. We do not know when mercury nitrate was first used to help in felting fur but we know that when the Huguenots fled from France after the Revocation of the Edict of Nantes in 1685, they carried with them the secret of mercurial carrot, as we call it, the French still call it *le secret. Hatters' shakes* and *mad as a hatter* are old terms for the mercurial tremors and psychoses of chronic poisoning as seen in this trade in every country.

Studies of mercurialism in the hatters' trade in this country began with one published by J. Addison Freeman of Orange, New Jersey, in 1860. He described some 100 hatters of Orange, who were victims of typical mercurial poisoning, but 15 years passed before the next record appeared, quite as shocking as the first. Nothing was done about it, little attention was paid, none by the medical world. Then in 1912, 34 years later, the public was startled by a report made by a lay organization, the National Civic Federation which had made a survey of the mercury-using trades in New Jersey and New York. Of course the cases of poisoning the survey brought to light were only the severe, conspicuous ones. Of these 94 were found,

among them men incapacitated by palsies, four with complete paralysis of a limb, many cases of polyneuritis, two with insanity.

Now the situation in the felt hat industry is completely changed. This historic trade, full of traditions and established habits, has at long last abandoned the use of mercurial carrot and substituted mixtures containing sulphuric and nitric acids, and hydrogen peroxide. The great reform was accomplished in a way typical of our country, not by law but by agreement between manufacturers and state and Federal Public Health Services, just as was the control of radium in industrial use and of tetraethyl lead. It is the only way a quick and effective reform can be brought about in several different states simultaneously. Thus an old and notorious source of industrial sickness has been abolished, we hope completely.

Foreign literature is full of cases of mercurialism traced to the use of mercury vacuum pumps in making electric light bulbs. Nowadays we use these very seldom, never in electric bulb manufacture. Yet mercury is still an important industrial element, especially in the production of dry batteries, in making thermometers and barometers, in extracting gold and silver from the richer ores and in the electrical industry. High frequency induction furnaces, the constant potential department, the manufacture of tungsten and molybdenum rods and wires, all call for the use of quicksilver. And now a new source of poisoning, sometimes acute and severe in form, has appeared, namely the use of organic compounds as fungicides. The dimethyl compound of mercury seems to be the one most often used here, but a Canadian report mentions diethyl mercury. The symptoms as described by English, German, and Canadian writers differ from those caused by the inorganic compounds. There is no mention of gingivitis or salivation, the central nervous system is chiefly involved, peripheral nerves, cerebellum, cerebral cortex, and the onset is usually rapid. The most striking cases come from Germany. Koelsch writes of 22 fatal cases he was called on to investigate during the war. These were in a chemical plant where mercury ethyl acetone and mercury ethyl bromide were produced for use as fungicides. He writes that the illness was characterized by profound cerebral involvement. One is reminded of the similar difference between the action of inorganic lead and of organic, tetraethyl lead.

Carbon Monoxide

FORTUNATELY for the industrial worker carbon monoxide poisoning has always been of even greater importance in ordinary life than in industry and has therefore always attracted the attention of the best brains in the medical profession. It has increased greatly in importance with the increase in automotive vehicles with their exhaust gases, while the problem of exygen lack in aviation has brought about studies which can be applied also to carbon monoxide asphyxia.

In this last war the enormous increase of aviation led to studies of the effects of CO and altitude on the visual thresholds of aviators. McFarland and his colleagues found that CO anoxia produces a loss of visual efficiency at an atmospheric concentration much lower than has been supposed. The American Standards Association's limits for maximum allowable concentration are: 100 p.p.m. for exposures not over eight hours daily and 400 p.p.m. for exposures not over one hour daily. These are based on the studies of Henderson and Haggard in connection with the problem of ventilating the Hudson River tunnels and are safe enough for ordinary conditions, we have always supposed. But McFarland found that a much lower concentration would affect sharpness of vision. Even inhaling the smoke of a few cigarettes might be enough. In industry one can imagine how so slight an effect on vision might cause a serious accident, if for instance the man affected were controlling a traveling crane.

The two most bothersome problems with regard to industrial CO poisoning are still with us, the possible sequelae of acute gassing and the question of chronic poisoning. With regard to the first, there is by now a mass of material on the damage to body tissues of all sorts caused by severe anoxia, especially if it is prolonged, and fortunately many of the cases are non-industrial in origin so there is no question of compen-

sation, and many have involved persons of the upper classes who had the best of medical care. As to whether chronic CO poisoning is a clinical entity there are still varying views. It is admitted by all that CO is not a cumulative poison for it is rapidly eliminated but there is evidence that repeated attacks of anoxia bring about an accumulation of effects. The experiments of Lewey and his colleagues during the war point to a cumulative effect on the brain by repeated short exposures to an atmosphere devoid of oxygen. The brains of animals which have recovered promptly and without any visible injury from such exposure have shown cell damage, first of reversible nature but later of permanent, if the anoxia was repeated. Lewey also exposed dogs to the standard concentration of 100 p.p.m. for five and a half hours a day, six days a week, for 11 weeks. The dogs seemed to continue in perfect health but as early as the second week electrocardiograph changes appeared and if they were killed, autopsy revealed degeneration of heart muscle fibres, hemorrhages and necroses. Some of the dogs showed disturbances of gait and of reflexes and histological changes were found in the central cortex and in the basal ganglia.

Phosphorus

ANOTHER old poison is phosphorus. It was deeply entrenched in the match industry for all the years between 1833 when Lorinser, an Austrian, described 22 cases of phossy jaw which had developed among match workers in the 12 years following the invention of strike-anywhere matches, and the year 1908 when European countries signed the Berne Convention by which they agreed to give up the manufacture and the importation of white phosphorus matches. In this country a very early case of typical phossy jaw was treated in the Massachusetts General Hospital in 1851, some 15 years after the manufacture of white phosphorus matches began in Springfield. This was a very severe case, ending after six months in death from toxemia. There was, however, no mention of phossy jaw in American medical literature from 1851 to 1910, when John Andrews published the report of an investigation he had made for the Federal Deparment of Commerce of American match factories. We had always been assured that our factories were so superior to the British and European that phossy jaw was unknown in our country, but Andrews brought to light over 150 cases, some of them quite as severe as any in foreign countries. As everyone knows, that report led to the passage of the Esch law, which placed a tax on white phosphorus matches and led to the substitution of the harmless sesquisulphide. The Diamond Match Company held the patent for this French discovery but generously threw open to the whole country the use of the process. So one notorious dangerous trade was rendered safe and we all thought that phossy jaw was a thing of the past. But eternal vigilance is the price of industrial safety.

A sudden and quite unexpected reappearance of severe phosphorus necrosis took place in 1923 when a new kind of fireworks was manufactured in three plants on the Atlantic seaboard and white phosphorus was one ingredient. The management was both ignorant and indifferent before the attention of physicians and state authorities was called to the situation. Fourteen workers, all women but one, had developed intensive necrosis of the jaw and two had died of septicemia. The period of exposure in these cases was unusually short for the exposure was very great.

That source in its turn was abolished, and years passed before we heard any more about phosphorus poisoning in industry. However, it has emerged again. In 1946 Heimann of the New York State Department of Labor found three cases of phosphorus necrosis of the jaw in men working in the production of red phosphorus, converting the yellow into the red allotrope. In spite of routine medical and dental examinations including dental x-rays, phosphorus necrosis of the jaw appeared after exposures of two years, eight years and 13 years. The man who had been exposed only two years recovered without deformity, the one who had worked eight years lost all his teeth and part of the left mandible, with some facial deformity; the case which was slowest in developing was much the most serious. This man lost a large portion of the palate bone and there were fistulous tracts leading from mouth to nose

and nasal sinuses. These were still active after 18 months. Heimann traces the trouble to the bubbles which form and burst from the water over the yellow phosphorus, for this must always be handled under water since it burns in the air.

Finally there is a still more recent source of trouble from this very troublesome element, although so far no human injury has been reported. This is the electro-thermal production of elemental phosphorus to be used in the preparation of phosphate fertilizer in the T.V.A. Fleming, Miller and Swayne of the T.V.A. have attempted to find the maximum allowable concentration of phosphorus by subcutaneous injection of animals, but such results are of little value in solving the problem of workers' protection.

Heimann's cases seem to show the practical impossibility of providing foolproof protection. These men had all had a dental examination within one month of the development of the necrosis. In the days when white phosphorus was still used for making matches the English took elaborate precautions in their factories. Workers were forbidden to keep a glass of water on the work bench, to chew gum or tobacco while at work or to eat an apple or put a finger in the mouth or pick their teeth. Strict cleanliness of benches and provision of fume removal were required of the management, yet phossy jaw continued to appear and apparently it always will so long as that subtle and relentless poison is used.

A few metallic poisons which were formerly little used or used in ways not endangering health have suddenly emerged with prominence and have begun to show ominous characteristics. The most important are aluminum and beryllium.

Aluminum

Recently Koelsch of Munich, for many years the foremost authority in Germany in this field, has sent me abstracts of German reports on the effect of aluminum dust, reports which were not accessible to us during the war and which are of interest not only in connection with aluminum workers but also with men working in mines which are dusted with aluminum for the prevention of silicosis. Koelsch sent me abstracts of four papers which describe what the authors consider a definite characteristic picture of aluminum dust poisoning in men working in an atmosphere of finely divided particles. The symptoms consist of short breath, lessened vital capacity, stabbing pains in the chest, slight cough and expectoration. The special feature all note is the youth of the victims (older men are less susceptible) the shortness of exposure, the rapid course of the disease, (the latter two in sharp contrast with silicosis) and the fact that the process may progress after exposure ceases. Two cases of pneumothorax are described, attributed to great increase of intra-alveolar pressure, the aluminum dust impeding expiration. Two autopsies are described, the findings in which agree strikingly: extensive interstitial pneumonia with induration and shrinking. The interstitial tissue had undergone hyaline-collagenous degeneration. One author speaks of a silvery, metallic sheen on the cut surface of the lung. According to Koelsch there was an abnormal dust exposure in these factories, caused by war-time conditions, the lack of proper exhausts, the obstructed windows, necessitated by the black-out.

Beryllium

The newest of the industrial poisons of practical and rapidly increasing importance is beryllium. For the description of its sources, uses and action I am indebted to Dr. Harriet Hardy for I have had no experience at all in this field. It appears that descriptions of pulmonary lesions following exposure of workmen to dust or fumes of beryllium compounds were published in Germany, Italy and Russia during the thirties. The most extensive discussion was that of Gelman of Moscow who wrote the section on beryllium for the I.L.O. Encyclopedia. Observing not only what he called "disseminated injuries to the lung" but irritation of the mucous membranes and the skin, he wrote that it was not easy to separate the action of beryllium fluorides from that of other fluorides. However, in Germany the picture in the lungs described by Gelman was found in workers exposed to beryllium compounds free from fluorine. The picture was one of disseminated sclerotic changes with x-ray findings resembling miliary tuberculosis. Clinically there was

cough, reduction of chest expansion, mild dyspnea, with disproportionately severe physical findings of pulmonary involvement.

The first report of an occupational disease in the beryllium industry in this country came from the Cleveland Clinic in 1943. These first American cases were reported by Van Ordstrand, Hughes and Carmody in men producing beryllium oxide from beryl ore. The authors called the affection chemical pneumonia. The following year saw a similar report from Pennsylvania, this time from an industry employing a fluorescent powder which contained beryllium carbonate and manganese silicate. Then the Cleveland group came back with an extensive report of an illness which they boldly called beryllium poisoning, and of which they described 38 cases with five deaths. Briefly summarized, the symptoms were those of chemical pneumonitis, cough, dyspnea, substernal pains, anorexia, loss of weight, increasing fatigue. The onset was insidious, lasting some three weeks, after which a characteristic, diffuse bilateral haziness appeared in x-ray pictures of the lungs. Removal from exposure and rest in bed with oxygen, as indicated by cyanosis, was followed by recovery, but if then the man returned to work there was a relapse and the fatal cases were in part due to pulmonary edema following too early exertion. The autopsy findings were those of bilateral, acute, organizing, atypical bronchopneumonia. All the Cleveland cases were in men exposed to beryllium oxide in processing beryl ore.

In 1946 Hardy and Tabershaw published a report of 17 cases of what they called "delayed chemical pneumonitis" in 14 women and three men working in the fluorescent lamp industry and using zinc manganese beryllium silicate. Here we have a different clinical picture, one of delayed onset, the symptoms appearing from six months to three years after the victim had left the workshop, then dramatic dyspnea, great loss of weight and a poor prognosis. Six of the 17 died and the other 11 were disabled from 18 months to over four years with only one known case of complete recovery. The pathologic lesions in the lung were those of an atypical granulomatous process replacing normal tissue, and similar lesions were found in liver, spleen and lymph nodes. A very strange feature of the Massachusetts experience is the discovery of three cases, two of them fatal, in individuals who were associated with, but never went into a plant making fluorescent lamps. These, in history and autopsy findings, seem to belong to the same group as the 17.

Since the publication of this report Dr. Hardy has had case records sent to her from seven different states, including Massachusetts. By April 1947, there were 40 cases of delayed chemical pneumonitis known to the Massachusetts Division of Occupational Hygiene in workers in the fluorescent lamp industry. Since some 1500 workers have been exposed, this would mean a case incidence of 2.5% but obviously that figure is far too low, based as it is on the more or less accidental discovery of fairly advanced cases.

The reported cases of beryllium poisoning therefore have come from plants producing and casting alloys, making and using fluorescent powders. No dependable reports have as yet been made of the amount of beryllium in the air of working places where the poisoning occurred. Chemical tests seems quite inadequate to reveal small amounts but a spectographic analysis, developed in the Kettering Laboratory in Cincinnati and in the Massachusetts Division of Industrial Hygiene, promises to give results.

The latest contribution in this field, January 1948, comes from Martland and his colleagues. They discuss four fatal cases, one acute, the others chronic, and include one which dates back to 1934 and has only now been recognized as a case of chronic beryllium poisoning in a worker in the fluorescent lamp industry. A most interesting addition to our knowledge is the proof of the presence of beryllium in the organs of two and in the skeleton of one of these two. This was shown by the spectographic method in the Kettering Laboratory.

The importance of this new poison is growing rapidly, for it is an excellent ingredient for alloys, which are light and resistant to strain, it is a good refractory for firebricks and is used not only for fluorescent lamps but for many other electric appliances. To anyone who, like me, can look back to the early days of industrial medicine, this story of beryllium poisoning

is an amazing thing. Here, in the space of some six or eight years a form of industrial poisoning which was highly dubious, involving only a small number of cases, not included under compensation laws except in full-coverage states, observed by a small number of physicians, was promptly made the subject of thorough study with full publicity. A conference was held in Boston at which the late Leroy Gardner of Saranac agreed to undertake animal studies with beryllium and last year a symposium was held in Saranac where the data were assembled from all over the country and discussed. When I look back on the days when we were urged not to mention such a thing as TNT poisoning lest we drive all workers out of the plants, I can hardly believe it is the same country.

Cadmium

CADMIUM is another industrial poison whose use has lately increased, but our knowledge of its action has been fairly extensive for some time. However, the increasing use for alloys and for electric plating and recently for the production of storage batteries has added to the data formerly acquired. According to Fairhall the danger of cadmium poisoning in the electroplating process is slight, it is in the application of heat to the plated surface with the production of the oxide that the danger comes. The occurrence of cadmium poisoning in smelters of ore containing this element was noted by the German, Stockhausen, as early as 1656 and thoroughly discussed by Tracinski of Upper Silesia in 1888. By 1946 Fairhall was able to summarize the histories of 59 reported cases of acute cadmium poisoning with a mortality of 15%. The action of the cadmium oxide is chiefly on the lungs, and resembles that of nitrous fumes, with constriction of the chest, increasing dyspnea, cough, nausea and abdominal pain, bronchopneumonia, cyanosis and pulmonary edema.

As for chronic cadmium poisoning there has been a wide spread scepticism but recently some French reports have come from plants producing storage batteries by the new method. A supposedly slow action of cadmium appeared after five to 14 years' exposure and began as obstinate pain in the lower back and legs which increased till walking became impossible. By x-ray, certain bones, chiefly scapula, ilium and femur, showed lines of pseudofracture. Anemia of the iron-deficiency type was the only other definite finding except of a yellow discoloration of the teeth. Hardy and Skinner reported five cases of what they suspected to be chronic cadmium poisoning in men exposed to concentrations of 0.6 to 6.8 mg. per 10 cu.m. The illness was subacute, with anorexia, nausea, vomiting, epigastric pain, fatigue, *dopeyness*, tooth decay, low hemoglobin values. The most recent work is that of Princi of the University of Colorado, who is studying workers in a cadmium smelter where the electrolytic method is used and the men are exposed to dust of the oxide. The concentration of cadmium oxide is sometimes as high as 31.3 mg. per 10 cu.m. He confirmed the French experience as to a yellow line on the teeth and also low hemoglobin values and low red cell count.

The experimental work of Prodan in 1932 is still the most valuable in that field, since it was an attempt to duplicate industrial exposure. Both the oxide and the sulphide were used and both are toxic, the latter less so because of its relative insolubility. The attack is chiefly on the respiratory tract causing a rapidly developing pulmonary edema, or a pneumonia, if the course is less rapid. A fibrotic thickening of the alveolar walls occurs, which is permanent. In animals these lasting lesions followed even brief exposure to the fumes. Liver and kidneys show fatty degeneration, both organs retaining much of the ingested cadmium.

Although not in the field of industrial toxicology one must not omit mention of the large number of cases of acute poisoning resulting from cadmium-plated eating utensils. According to Fairhall up to 1941 only 21 such accidents were reported, but since then no less than 268.

A curious fact noted by Church and Princi is worthy of mention. This is the apparent antagonistic action of selenium on cadmium as shown experimentally and confirmed in practice. In one cadmium smelter the workers were free from any sign of poisoning although the concentration of cadmium in the air was up to 2000 or 3000 times the maximum allowable con-

centration. The air of this plant has small traces of selenium.

Chrome

THE local action of chromic acid droplets and chromate dust is very familiar to workmen and has been for over a century. Chrome ulcers, chrome holes, perforated nasal septa are certainly troublesome but never really disabling and we still feel certain that such lesions do not undergo cancerous change, for they have been under observation for over a century. But lately reports have been coming from Germany of cases of pulmonary carcinoma in men exposed to chromate dust, not many cases, it is true, but numerous enough to make such authorities as Koelsch and Teleky believe that there is a causative relation. So far no such reports have been published in this country, but of course we must expect the same experience, that is observed in other countries invariably turns up over here. That the cases so far reported all occurred in men exposed to chromate dust, not to droplets of chromic acid, is probably due to the greater precautions taken against the very irritating action of the acid.

Benzene

TURNING now to the organic compounds, no industrial poison except lead and carbon monoxide has had the amount of scientific study devoted to it that has coal tar benzene, because it is so valuable a solvent and at the same time so dangerous to use. The history of the coal tar solvents, benzene, methyl benzene which is toluene, and dimethyl benzene or xylene, is fairly recent in the United States. Before 1914 we did not produce these solvents from coke. The valuable by-products of coke furnaces simply escaped into the air and we bought what benzene was needed from Germany. Then came the war and the blockade, the demand from the allies for explosives which require benzene, which was used in the rubber industry and in the hastily started aniline dye industry. So coke by-products plants were built and suddenly there was a wide spread use of coal tar solvents and their derivatives. It was only to be expected that cases of industrial poisoning began to appear, for medical men knew nothing of the effects to be looked for and sanitary engineers had no idea of the danger points to be avoided. I remember a worried du Pont chemist telling me he could not understand why a worker should be fatally poisoned when there was no more than 5% of benzol in the air where he worked.

The study made by the National Safety Council under C-E. A. Winslow started us on the right road and soon American laboratory studies and observations in the field widened our understanding and led to a marked fall in the incidence of poisoning. But the greatest advance was made when latex came in as a substitute for the various solutions of rubber. Here, as in the case of white phosphorus matches and of carbon disulphide vulcanization and of mercury-free felt hat carrot, a safe substance takes the place of a poison. Latex is the gum of the rubber tree and instead of coagulating it and then dissolving it, for dipped, spread and cemented rubber goods, it is shipped as a liquid in tankers and used as a liquid.

Unfortunately benzene has many other uses for which latex is not a substitute and it requires unremitting vigilance on the part of the industrial physician if he would avoid trouble. The more recent studies, especially those made by the Boston and New York groups, of workers exposed to benzene have shown that the earliest sign of intoxication is not always, as we used to believe, a drop in the white cell count or even a low polymorphonuclear count, but it may be a drop in the red cell count with the appearance of large erythrocytes, a macrocytic anemia. Another important addition to our understanding of the action of benzene we owe to these Boston and New York studies. No longer do we believe that the attack of benzene is on one element only of the bone marrow, rather we insist that any abnormality in the blood picture, if it appears in a benzene worker, must be treated as a warning of danger. That is the only safe course to follow.

Still another important finding is the fact that marrow aplasia is not an invariable finding in severe benzol poisoning, indeed hyperplasia is the characteristic finding in many cases. These observers suggest that the reason why we have believed women to be much more susceptible to benzene than men may be that cases with hyperplastic

marrow were rejected, and it is true that one can find in the older literature instances of such a decision. My experience with compensation boards bears this out. Nevertheless I still believe that benzene is especially dangerous to menstruating women.

The entrance of the methyl radical removes from toluene and xylene this special action of benzene on the bone marrow and several extensive investigations made during the war serve to confirm the comparatively harmless nature of these two solvents.

As to the benzene derivatives which have found a place in industry, their name is legion and our knowledge of their action in many cases is founded only on animal experiments. Even more numerous and more complicated are the derivatives of the petroleum series, the chlorinated hydrocarbons, so valuable because they are non-inflamable, a group of which the chlornaphthalenes are the newest members; the glycols, which are bivalent alcohols; the cyclohexanes and hexanols; dioxan, which is diethylene dioxide. These are all of increasing importance and call for continued observation in practical use.

Methanol

FORTUNATELY methyl alcohol is no longer a cause for apprehension. Our history in regard to that poison is unique and not very creditable. First, under the influence of the wood alcohol producers and the total abstinence advocates, we forbid the use of revenue-free grain alcohol, so that the use of wood alcohol was strongly entrenched in American industry at the time when all other industrial nations used denaturated grain alcohol. Then when Prohibition came in and reports of blindness and death from bootleg liquor shocked the public, we went to the other extreme, forbidding even the addition of wood alcohol to industrial grain alcohol as one of the denaturants. Wood alcohol still carries its bad reputation and when it is used, elaborate precautions are taken, precautions one would like to see applied to more dangerous liquids.

Carbon Disulphide

CARBON disulphide is an industrial poison which was for many years curiously neglected by the American medical profession. I came across it first in 1914 when I was studying the rubber industry. The German, the Italian and the French literature were full of discussions of this poison to the central nervous system, for carbon disulphide was an essential element in the rubber industry in those countries. England did not use it as much and we Americans still less. But we did at that time use it for the vulcanization of thin rubber goods and I discovered cases enough in Akron. Not through physicians, who seemed never to have heard of it, always only through foremen. Yet the cases of sudden maniacal attacks and then of more slowly increasing motor paralysis were dramatic enough to attract attention.

Fortunately this vulcanizing agent was given up by the rubber people soon after the First World War, but then it came back in greater force when viscose rayon appeared and it spread far more widely than it ever had in the rubber trade. Yet as late as 1938 this industrial poison was practically ignored by American doctors. I found that the insane asylums to which the victims were sent, recorded the cases with extreme caution as due to "some exogenous poison" or "etiology unknown" or "possibly occupational in origin." In those rare instances when compensation was sought, it was apparently always possible to secure the services of a professor of neurology or of a psychiatrist to testify that there was no such thing as carbon disulphide intoxication. The only explanation for this ostrich-like attitude is that the plants were largely situated in states with very poor factory inspection departments and still poorer compensation systems.

Carbon disulphide is still essential in the production of viscose rayon but now the plants which used to ignore the danger are admirably safeguarded and routine medical examination serves to detect the trouble at an early stage and remove the victim from further exposure. Trouble there will always be so long as carbon disulphide is used, for there is no way of detecting in advance which individuals will prove to be oversusceptible. One good feature of the poison is that both the central and the peripheral nerve injury are reversible. The tragically severe cases which we found 10

years ago were usually in men who were allowed to go back to their former jobs and of course that would never happen now.

Synthetic Rubber

ONE of the newer industries is the making of synthetic rubber which was of very great importance during the war and is now firmly established. Fortunately it was developed so recently that it had no bad practices inherited from the past and from its inception the industry has been much under the eyes of competent physicians and sanitary engineers so that the risks and the way to control the risks have been studied thoroughly. Of course there is no telling when our extremely capable and inventive industrial chemists will introduce new compounds, indeed already the list of organic accelerators and anti-oxidants is formidable, but the solvents are mostly familiar to us, the coal tar solvents and the chlorinated hydrocarbons.

According to Mallette of the Firestone Company there are five principal types of artificial rubber manufactured in the United States. The toxic materials which are new are not many and few of them present a serious danger. The following have been tested on animals and on men: acrylonitrile, which is an organic cyanide, vinyl cyanide, and has the well-known cyan action; butadiene, a mild narcotic with an irritating effect on the eyes and the air passages, sometimes affecting the lungs, but with no cumulative action; monomeric styrene which is more productive of discomfort to human beings than either butadiene or toluene. The irritating effect on eyes and nose is supposed to give ample warning of danger, a rule which sad experience with other little known poisons has proved to be unreliable. In animals, heavy exposure to monomeric styrene causes immediate death from a primary action on the central nervous system, delayed death from pneumonia. Such a finding means that a job of repairing or cleaning apparatus may be fraught with great danger. The fourth one is chlorobutadiene, known as chloroprene. It has been found to be decidedly toxic to animals, causing depression of the nervous system, a low blood pressure and severe degenerative changes in the organs.

Then there is a long list of old familiar poisons used as ingredients or as solvents for synthetic rubber, besides those already mentioned.

Prevention

IT IS now an accepted duty of the industrial physician to examine for employment all new men and assign them to various kinds of work according to their physical condition, but such examination will never weed out the over-susceptible. Only a close watch over the newly employed during the early months will do that for, as is true of the infectious diseases, the victim of lead or mercury or nitrous fumes may be a physically perfect specimen whose idiosyncrasy cannot be detected in advance. Frequent re-examination during the first months is the only way the physician can detect and remove from exposure such a worker.

This brings us to the question of alternative workers in dangerous jobs. When I was in England studying the storage battery industry, I found that they laid great stress on this method of preventing plumbism. We have never used it here to any extent. The workmen object to it, understandably. Of course we do remove a man from a toxic job but we do not send him back to it after a spell at other work. Moreover we have been told recently by the du Pont physicians that they have given up removing from the job men who show, on cystoscopic examination, a condition leading to what we still call aniline tumor of the bladder. This is because they found that the process keeps on after exposure ceases, unless properly treated. They leave the man at work under treatment.

Although no measures of personal hygiene will save a worker from occupational disease if the conditions of work are bad, yet there are certain rules of hygiene that are decidedly useful. Both human observation and animal experiments have shown that the presence of milk in the stomach inhibits to a decided degree the absorption of lead. British and German employers have for half a century had the habit of providing milk for lead workers the first thing in the morning or in the middle of the morning and afternoon. Studies of carbon tetrachloride therapy for hookworm infestation have shown that a negative calcium balance from lack of calcium in the

food encourages a toxic reaction, also that it is possible to defend the liver from damage by a diet rich in sulphur-containing proteids. German and Russian writers hold that the obese are more susceptible to benzene than the thin and that fatty food favors poisoning. This was confirmed by the experiments of Li and Freeman who showed that a diet low in proteins and high in fat increased the action of benzene on rats and on dogs. The effect of alcoholism is notoriously bad on poisoning from lead, mercury and TNT, and pharmacologists found that to be true of carbon tetrachloride also.

Foulger, Director of the Haskell Laboratory of the du Pont de Nemours Company, emphasizes the importance of proper food for workers in the poisonous trades. He has found that such workers need an optimal content of vitamin in their daily food, particularly of vitamin C and the B group. It may be simpler and more effective to administer these vitamins than to try to control the eating habits of the men and women in these jobs. Foulger quotes Viscount Chetwynd, manager of a TNT filling factory in the First World War, as saying that the women in the factory had far more gastric disturbances than the men. On the theory that this was caused by the less substantial meals eaten by the women, the management made a deduction from their pay and fed them well. As a result, the complaints fell off from 11.3% in September to 0.7-2.0% the following January. Foulger says that the night shift needs special attention in this respect.

May I close with a few warnings:

1) The establishment of so-called maximum allowable concentrations by the A.S.A. for industrial poisons has given us valuable guidance, but do not forget that these are based on the quantity inhaled by a man breathing at a normal rate for eight hours. Over-time and heavy, hot work, making the man breathe more deeply and rapidly, make necessary a lower standard.

2) Statements as to the toxicity of an element must be taken with caution for there may be a wide difference in the different compounds. The basic carbonate of lead is much more soluble in the body juices than the straight carbonate; the sulphate is fairly soluble, the sulphide very slightly so. We look upon zinc compounds as only slightly toxic, but wartime experience here and in England showed that zinc chloride in the form of fumes can be deadly.

3) Be cautious in applying the results of animal experiments to human beings. We were misled for years by accepting the picture of benzene poisoning as seen in animals where leucopenia is the typical sign, appears early and is a safe warning of intoxication, anemia is slight or absent. So for years we depended on the white cell count only and missed many cases. In carbon tetrachloride poisoning in animals, it is the attack on the liver that is conspicuous, not the kidneys, in industrial poisoning the reverse is true. Animal experiments are of course very valuable but they can never do away with the need of close observation of human beings. They would never have helped us to detect early mercury poisoning, with its increasing timidity and depression, or early carbon disulphide poisoning, with its irritability and gloom.

4) In making your diagnosis do not follow too scrupulously the typical picture as given in the text-books, those books with which you will be confronted when you go before the Compensation Board. It is only in industrial cases with their unfortunate medico-legal side that such a strict demarcation line exists. The literature on blood dyscrasias is full of atypical cases but we have only lately been able to discard the typical picture in benzene poisoning.

5) Be cautious in accepting the statement of the employer as to the compounds to which your patient was exposed. Often he does not know what a trade name means—there are more than a dozen strange names for carbon tetrachloride—or he may think he is still using tetrachlorethylene as a solvent when the purveyor has without warning switched over to tetrachlorethane. Or the straight-run gasoline he formerly used has been changed to "cracked gasoline" which contains benzene.

6) Finally we must take with caution the statement that experiments with a certain new poison show that sufficient warning of danger will be given in time, by an irritating action on the eye, nose and throat. That may be true when employment is high and compensation laws adequate. It cannot be depended on when jobs are scarce or in states with poor labor laws.

Effect on Noise and Light of Cleaning and Painting Acoustical Ceiling

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THIS study was undertaken to evaluate the effect of cleaning and painting on the performance characteristics of acoustical ceilings, and to ascertain potential improvements in the light intensities of a work room area by increasing the reflection factor of the ceilings, cleaning the reflectors and replacing the lamps before the end of their useful life.

Introduction

THE general trend in recent years has been to increase the lighting intensity (footcandles) on the working plane. For example, in a pamphlet issued by the Illuminating Engineering Society in September, 1942,[1] it is recommended that a minimum of 25 footcandles be provided for ordinary seeing tasks (a) discrimination of moderately fine detail, (b) better than average contrast, and (c) intermittent periods of time. A report of the Office Lighting Committee of the Illuminating Engineering Society, published in 1947,[2] recommends 30 footcandles for the seeing tasks referred to above. Consequently, during a five-year period this Society has deemed it desirable to increase the illumination by 5 footcandles for similar work.

Noise has been defined as any unpleasant sound, the elimination of which is desirable. Noise is a sound that forces unwilling attention, causes an unpleasant emotional reaction, and gives a person a distinct feeling of relief upon cessation. The field of industrial hygiene is interested in noise since there is both practical and experimental evidence to indicate that noise produces fatigue, impaired hearing, neuroses, decreased efficiency, and emotional disturbances.[3]

Numerous articles have appeared on the loudness of various noises as measured by different investigators. There is, however, very little information regarding optium noise levels. KNUDSEN[4] states that the maximum noise level desired in private offices ranges from 20-30 decibels. It should be mentioned that this level is suggested but is very seldom attained in practice. It is, therefore, desirable in a private office to maintain a minimum of 30 footcandles for ordinary seeing tasks and a maximum noise level of 30 decibels.

The authors feel that leading architects are striving to meet these standards. Acoustical ceilings are not unusual in office buildings and luminaires are constantly being improved to provide adequate illumination. However, a factor often overlooked is maintenance. New office buildings usually meet the requirements for good lighting and noise levels, but after several years it is found that these standards are no longer met.

In this investigation, the authors made detailed illumination and acoustical studies of four different rooms. Acoustic tile had been used for the ceiling of each of the rooms; the luminaires were of the indirect type.

With such luminaires 90-100% of the light is first directed to the ceiling and upper side walls from which it is diffusely reflected to all parts of the room. In effect, the entire ceiling becomes the light source, and the shadows and the reflected glare are minimized. The ceilings of the test rooms had not been refinished for several years and as a result were badly discolored.

Investigation

THE objectives of the investigation were two-fold:

1. An evaluation of the improvement in illumination, and
2. An evaluation of changes in the acoustical qualities of the ceilings.

In the improvement of illumination, three factors were considered:

1. The painting of the ceilings.
2. The cleaning of the reflectors.
3. The replacement of the lamps before the end of their useful life.

Painting the Ceilings

THE ceilings were lined with a fissured type acoustical tile. To prepare the acoustical tile surfaces for painting, the ceilings were thoroughly cleaned of all surface dust and dirt by the use of a vacuum cleaner with brush attachments. In addition, all surfaces were hand-brushed or wiped clean of dust and dirt. The ceilings were then given one coat of resin emulsion paint in accordance with Federal Specification TT-P-88-a,[5] white color. Spots not removable were painted over sufficiently to match other painting. The paint was applied with a spray gun with the exception of the edges of the ceilings which were brush-painted.

Cleaning Reflectors

THE effect of glare and high brightness ratios and their relationship to symptoms such as headaches, eye-aches, blurred vision, and fatigue, are well known and will not be discussed in this article. The majority of architects recognize these factors in designing lighting for office buildings and frequently use indirect lighting to minimize glare and high brightness ratios. However, indirect lighting fixtures with inverted bowls are inherently difficult to keep clean. Failure to keep the reflectors clean results in a progressive diminution of the lighting intensity on the working plane, sometimes far below the designed illumination level.

In this investigation the reflectors were cleaned with soap and water. This operation consumed approximately five minutes for each reflector.

Replacing Lamps

THE light output decreases as the filament of the lamp slowly sublimes, becomes thinner, and thus less power is consumed. Moreover, the filament is at a lower temperature, and there also may be considerable bulb blackening. It is economically sound to remove lamps from service whenever the cost of energy consumed per million lumen-hours exceeds the average cost of life up to that time, including both lamps and energy.

Lamp economics involves in addition to lamp and energy costs the third element, replacement.[6] In large buildings such as

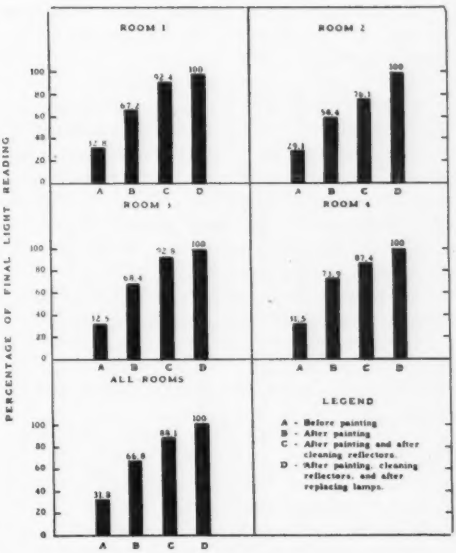

Fig. 1
Percentage of final light reading (A) before painting, (B) after painting, (C) after cleaning reflectors and (D) after lamps were replaced respectively

office buildings the cost in many cases of replacing individual lamps as they fail is relatively high. Under these circumstances the group-replacement plan is particularly valuable. At 90% life, 72% of the lamps are still burning, at 80% life 84% remain. Replacement of 70% average life is often times adopted as the economical point for group replacement, for at this point less than 10% of the lamps should have normally failed. The most economical schedule will differ in accordance with conditions and requirements for each specific installation.

In the investigation all of the lamps were replaced in the rooms studied regardless of how long they had been in use. The lamps replaced were in various stages of their useful life; some were quite dark while others were practically new.

Determination of Illumination

ILLUMINATION readings were taken 30 inches from the floor or at desk level with a Weston Light Meter, model 614, using a Viscor filter. Sufficient readings were taken in each of the rooms to give adequate

data. Blinds were drawn over the windows in each of the rooms investigated to minimize the effects of natural illumination.

Determination of Sound

THE effect of painting on the acoustical qualities of the ceiling was evaluated with a General Radio Sound Level Meter and Frequency Analyzer. The readings were taken with the microphone placed on a tripod. Extreme care was exercised to locate the microphone in the same position in the room for before-and-after readings. Sufficient readings were taken to give adequate data on each of the rooms. Most of the noise emanated from forced draft air conditioning units located in each of the rooms.

Results

THE process of cleaning and painting the acoustical ceiling material in the rooms studied resulted in an increase of the average reflection factor from 25 to 70%.

Results of the investigation are shown in Table 1, and Figs. 1 and 2. Table 1 contains the average light intensities for each room before any improvement and after each step in the improvement of illumination. In column A of the table it may be seen that the light intensities before painting varied from 6.1 to 8.2 footcandles, with an average for all rooms of 7.2 footcandles. The light intensities after painting the ceilings are shown in column B. The average for all rooms was 15.1 footcandles or 2.1 times the initial intensity.

TABLE 1
AVERAGE LIGHT INTENSITIES OF ROOMS IN STUDY WITH COMPUTED RATIOS

	A	B	C	D	D/C	D/B	D/A
1	8.2	16.8	23.1	25.0	1.08	1.49	3.05
2	6.1	12.2	15.9	20.9	1.32	1.71	3.43
3	6.8	14.3	19.4	20.9	1.08	1.46	3.07
4	7.0	16.4	19.4	22.2	1.14	1.35	3.17
All rooms	7.2	15.1	19.9	22.6	1.14	1.50	3.14

A—before painting, B—after painting, C—after reflectors cleaned, D—after lamps replaced.

The value for all rooms as indicated in column C is 19.9 footcandles, the average intensity after the reflectors had been cleaned. This is an increase of 31% over the intensities after painting, and 2.76 times the intensities before painting. Column D shows the final light intensities obtained; the average for all rooms was 22.6 footcandles.

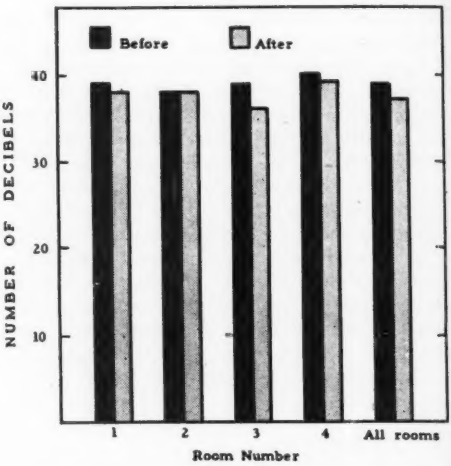

Fig. 2
Effect of cleaning and spray painting on the acoustical qualities of soundproof ceilings

The increase in illumination from column C to D resulted from replacement of all the lamps in the fixtures regardless of their length of service. It may be seen that Room 2 had the greatest increase of all rooms, an increase of 5 footcandles or 32%. The lamps removed from the fixtures in this room were very dark and evidently near the end of their useful life.

In Table 1 the ratios of the final light intensities to the initial and to the other intermediate steps have been computed. The Table indicates that the intensity of light in footcandles after improvements had been made was 3.14 times the intensity initially found or a 214% increase for all rooms. Similarly, the final readings (average for all rooms) were 1.5 times the readings obtained after painting. This clearly shows the necessity of maintaining surfaces with a high reflection factor both with respect to the reflectors and to the ceilings of the room.

Fig. 1 shows graphically the improvement in illumination for each room and the average of all rooms. The light intensities found initially and after each step in the study were determined as a percentage of the final intensity taken as unity. For example, in the graph for all rooms in

Fig. 1 bar D represents 100% of the total light intensity, or 22.6 footcandles. Bar A represents 31.8% which is the initial light intensity found, or 7.2 footcandles. Bar B indicates the percentage after painting, or 15.1 footcandles. Finally, bar C shows the percentage after painting and cleaning of reflectors, or 19.9 footcandles.

As shown in Fig. 1, Room 2 differs from the other rooms with lower values in bars A, B, and C, and a larger increase from C to D. This is due to the fact already mentioned that for this room the increase in light intensity by replacing lamps was 5 footcandles while for the three other rooms it averaged 2.1 footcandles. It will be remembered from above that in room 2 the lamps were very dark and were evidently near the end of their useful life.

Fig. 2 shows graphically the results of the study on the effect of cleaning and spray painting the acoustical material on the ceilings of the rooms. It may be seen that in each room the number of decibels after cleaning and painting was the same or slightly less than before these operations.

Conclusion

THE study demonstrates (1) that acoustical ceilings can be painted without affecting the acoustic qualities, (2) that when indirect lighting fixtures are used the ceiling must be maintained with a high reflection factor, (3) that a considerable improvement in lighting intensity can be obtained by maintaining the reflectors in a clean condition, and (4) that lamps nearing the end of their useful life cause a marked decrease in light intensities (footcandles) on the working plane.

In this investigation the average light intensity was increased from the initial value of 7.2 footcandles to 22.6 footcandles or an increase of 214%. This graphically illustrates that in many cases light intensities in a work room area can be increased without the necessity of expensive new installations or alterations.

References

1. Committee on Lighting Practice of Illuminating Engineering Society: Recommended Practice of Office Lighting. September, 1942.

2. Office Lighting Committee of Illuminating Engineering Society: Recommended Practice of Office Lighting. *Illuminating Engineering*, 42, 673 (July) 1947.

3. MCCORD, C. P., TEAL, E. E., and WITHERIDGE, W. H.: Noise and its Effect on Human Beings. *J.A.M.A.*, 110, 1553 (May 7) 1938.

4. KNUDSEN, V. P.: Architectural Acoustics. John Wiley and Sons, New York, 1932.

5. Federal Specifications for Paint: Resin-base emulsion, Interior, Paste, White and tints. Federal Standard Stock Catalog, Sec. IV (Part 5). TT-P-88a. U. S. Government Printing Office, Washington, D. C. (May 26) 1945.

6. General Electric Engineering Division, Lamp Department: Lamp Bulletin LD-1. Cleveland, (May) 1946.

PERSONNEL PLACEMENT COMMITTEE ACTIVITIES

THE Personnel Placement Committee composed of FRED R. INGRAM, Chairman, W. G. FREDRICK, and CLYDE M. BERRY, have accomplished much over the past year in bringing together industrial hygiene personnel and agencies interested in their services. The following table gives the number of referrals.

	Agencies	Personnel	Total
May 1, 1947—October 31, 1947	18	17	35
November 1, 1947—March 31, 1948			
Industrial Hygiene Engineers	8	8	16
Industrial Hygiene Chemists	8	5	13
Industrial Hygiene Physicians	9	2	11
Physiologists and Toxicologists	1	0	1
Industrial Hygienists	6	0	6
Dentists	0	1	1
TOTAL	32	16	48
TOTAL FOR 1947-48	50	33	83

The Case for Maximum Allowable Concentrations

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SINCE the publication in 1927, in the International Critical Tables, of data on physiological response to various concentrations of gases and vapors, including concentrations allowable for prolonged periods, the subject of maximum allowable concentrations (MAC's) has received much attention from persons interested in protecting the health of workers. Some of the authorities who have contributed in this field are SAYERS, DALLAVALLE, PHILIP DRINKER, BOWDITCH, YANT, STERNER, TELEKY, LEHMAN-FLURY, FREDRICK, FAIRHALL and last but not least WARREN COOK.

The organizations interested in MAC's have included the U.S. Public Health Service, most of the state and local bureaus of industrial hygiene, the American Standards Association and the American Conference of Governmental Industrial Hygienists. Data have appeared in books on industrial toxicology, industrial hygiene, industrial health engineering, the care of the handicapped worker, and various hand books. Certainly any proposition with such substantial backing must be sound, and acceptable to all interested in industrial health problems.

Yet there has always been a small minority which has been skeptical of, and even hostile to, the use of MAC's in preventing occupational disease. Their criticisms vary all the way from complaints about the term "MAC" to personal criticism of the personalities and groups associated with the promulgation of MAC values. Nor does this group consist entirely of obstructionists and professional objectors. At the last meeting of the New England Section of this Association, three of our most eminent and respected members took occasion, out of a clear sky, to decry one phase or another of MAC's.

One speaker, an engineer, stated that there was too much emphasis on MAC's, leaving the inference, at least, that more attention should be paid to the engineering aspects of control. Another speaker, a physician, deprecated the use of MAC's in place of medical control measures, implying that the latter alone were sufficient. The third speaker, while not condemning MAC's in general, warned dramatically against suggesting values based on insufficient data. As he put it, we should not "draw a value out of thin air."

Presented at the Ninth Annual Meeting of the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION, April 1, 1948, Boston, Massachusetts.

Fundamentals of MAC Selection

I SHOULD like to consider these criticisms and their implications. First, however, it might be worth while to review a few fundamentals. The incidence of intoxication, with different exposures, is shown by the familiar probability curve. Horizontally the minimum concentration which will cause intoxication is given, while vertically we have the number of persons affected. This is merely a graphical method of expressing variations in individual susceptibility. (Fig. 1.)

At very low concentration no one is injured; as we increase the exposure a level is reached where a very few individuals are intoxicated; with a further increase the number of additional persons affected per increment of increase becomes greater until a concentration is reached where approximately half the population has succumbed. Then each further increase in concentration affects a diminishing number of additional people until all are intoxicated, when further increase is naturally without effect.

At what point on this curve should we select the MAC? The middle of the curve, the LC_{50}, if our criterion of intoxication is death, otherwise the TC_{50}, is the value which the experimental toxicologists quote. Even the most backward industrialist, however, would not recommend the TC_{50} as the MAC. If the effects of intoxication are permanent, death or disability, or even serious illness, the MAC should be below the value which produces these effects in even highly susceptible individuals.

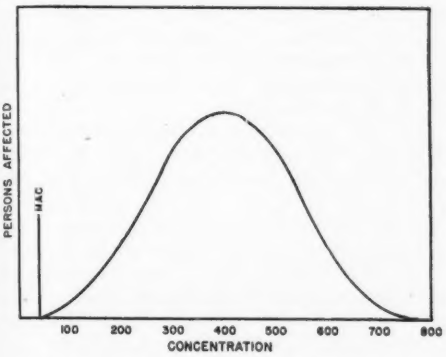

Fig. 1
Probability Curve of Percentage of Persons Affected by Increasing Concentrations of Toxic Substances. (Percentages not Cumulative)

On the other hand, if the illness is rather minor and transient, the MAC can be chosen at a level which does affect a minority of workers, but should be below the value which produces these effects in eve nhighly susceptible individuals.

On the other hand, if the illness is rathei minor and transient, the MAC can be chosen at a level which does affect a minority of workers, but should be below the value where the average individual suffers.

Some people hold that these minor illnesses should be ignored in establishing MAC's. Thus the lachrymation due to formaldehyde, the cough and throat irritation from sulfur dioxide, the runny nose caused by fluorides, the headache resulting from absorption of nitroglycerin, and the fever attributable to zinc fumes, should not be considered as justification for lowering MAC values for these substances to a point where the symptoms noted do not appear.

Now we have heard much recently of the importance of the common cold as an industrial hygiene problem, and ambitious programs to eliminate the common cold from industrial workers have been advocated. To the best of my knowledge these programs, in principle at least, have met with little opposition.

What are the symptoms of a common cold? Your eyes water, your nose runs, you cough, you have a headache and a slight fever—exactly the symptoms produced by so-called non-toxic concentrations of the gases and fumes just mentioned. Yet the same people who are unwilling to ask industry to spend a few dollars to eliminate these symptoms, when they are obviously occupational in origin, are proposing that vastly larger sums be spent in measures of, as yet, unknown efficacy to eliminate the same ill effects when caused by infections not directly attributable to the employment.

What About the Criticisms of MAC's?

THE first one was that MAC's are overemphasized, at the expense of engineering principles. We should spend less time comparing the concentrations of dusts and gases in the work room with the MAC, and more on streamlining exhaust systems and checking air flows. Engineers forget, when they present this argument, that good engineering, when involving toxic dusts and fumes, is a means and not an end. Which is more important to the health of the worker—that the quantity of lead dust, or carbon tetrachloride vapor, be kept at a harmless level, or that the air flow through the exhaust system comes up to the standards of good engineering practice?

From the standpoint of purely engineering considerations benzene vapor is practically identical to the vapor of hexane, methyl bromide and ethyl chloride are almost the same, as are the fumes of zinc oxide and cadmium oxide, and the dusts of calcium carbonate and radium carbonate. If control measures are based solely on engineering factors, either the worker will not be protected, in the case of the toxic material, or else the employer may be penalized, if the substance is non-toxic. If the toxicities of the contaminants are taken into account in specifying control measures, some kind of MAC values must be used.

We have in this state several storage battery plants. The lead hazard inherent in such establishments is familiar to all of you. One of these factories grew, like Topsy, from the owner's basement shop into a building which was formerly a garage, which has since been expanded by adding a little here and a little there. As can be imagined, this plant leaves much to be desired from the standpoint of the engineering perfectionist.

Two of our other plants were planned from scratch by men who knew the business. Each has been characterized by well

known engineers as an ideal plant. But lead dust concentrations were found to be much higher in the ideally engineered plants than in the Topsy-like plant first mentioned. And the incidence of lead poisoning, strangely enough, seems more closely related to the concentration of lead dust in the air than to the degree of streamlining of the exhaust system.

It would appear that in many cases the emphasis is not an MAC's at the expense of engineering standards—rather the shoe is on the other foot.

How about the second criticism, that we should rely more on medical controls than on MAC's? It seems strange that this opinion should be held today, when the entire philosophy of industrial hygiene is prevention of ill health. It is well recognized that in many cases medical science is unable to detect incipient poisoning until it is so far advanced that permanent injury is probable.

Let us consider just one incident. In a paper by A. R. SMITH in 1945, a delayed case of benzene poisoning is discussed. The worker, when first examined, during or just after a period of exposure, had a white blood cell count just below normal. Three months later, with no further exposure, his blood picture was normal. But four years later he died, and the diagnosis was benzene poisoning.

DR. SMITH identified for me the plant in which this man worked. Air analyses showed benzene vapor concentration up to 300 p.p.m., and averaging 115 p.p.m.—clearly a severe benzene hazard. Yet if we relied solely on the medical examination of a worker so badly poisoned he was to die, we could only say there was a possible hazard, a borderline case.

To abandon MAC's and rely solely on medical control measures would be to take a long step toward the dark ages of empiricism in occupational disease control.

Finally, what about MAC's taken out of thin air? At first glance it would seem that this is a reprehensible practice, which should be sternly condemned. What, however, are we to do when confronted with the industrial use of a toxic substance for which data on which to base an MAC are lacking? We have several alternatives: (1) no controls, (2) controls based solely on good engineering practices, (3) medical examinations, and (4) fume control based on a tentative MAC.

Let us consider an example: The Massachusetts Fume Code Committee, in 1938, included tetrachloroethane in its list of vapors for which MAC's were proposed. There was no information on industrial exposures, and animal data were inadequate to lead to an MAC value. The committee arrived at a figure in a round-about fashion. Certain German authors had stated that tetrachloroethane was 10 times as toxic as carbon tetrachloride. Since the effects of these two vapors were roughly similar, and the MAC for carbon tetrachloride was then considered to be 100 p.p.m., a value 1/10 as high, 10 p.p.m. was proposed for tetrachloroethane.

Surely this procedure can be classed as "taking a value out of thin air." That was certainly the opinion of the American Standards Association committee and various other authorities, who berated us for proposing such an absurdly low figure.

During the war tetrachloroethane was used in some quantity in a process which need not be described here. I am indebted to Mr. Halpin of the Army Industrial Hygiene Laboratory for information on the experience of the Army with this solvent. I quote from his letter as follows: "Numerous cases of jaundice and toxic hepatitis were reported among workers exposed to tetrachloroethane. Many atmospheric analyses revealed concentrations of less than 10 p.p.m. at the time of sampling, yet clinical and sub-clinical effects continued to be found."

From this evidence it would seem that 10 p.p.m. is too high for the MAC of tetrachloroethane. If these illnesses were actually due to the use of an MAC "drawn out of thin air," it is indeed a serious indictment against the practice of proposing MAC values based on meager data. However, is there any evidence that the existence of the MAC value contributed to these cases? Would there have been better control if there had been no MAC? I doubt it very much.

The experience of the Army with illness from mustard gas in shell filling plants

might be pertinent to this matter. No MAC for mustard gas had been proposed, so engineering and medical control measures were relied upon with no MAC's to becloud the issue.

What happened? I will quote from the Industrial Hygiene News Letter of January, 1948. "About a thousand workers in Huntsville Arsenal. . . .show symptoms of excessive exposure to mustard gas. . . .About 300 are under observation or treatment. . . . Most have a true physical disability; a psychoneurotic component is evident in many."

From these cases one can infer that the use of too high an MAC, as opposed to no MAC at all, does not increase the incidence of occupational illness. However, it does prevent the extreme effects which would result from very heavy exposures, which might otherwise occur. In other words, we can argue that half a loaf is better than none.

There is one clear advantage to use of a tentative MAC drawn out of thin air. We now know that the correct MAC for tetrachloroethane is below 10 p.p.m.—probably study of the Army's data above cited will tell us the proper figure. And where do we stand with mustard gas? So far as available information goes, we are exactly where we were 20 years ago as far as knowing what a safe consideration is.

Establishment of a tentative MAC, from thin air or otherwise, is only a first step in determination of proper and necessary control measures. It encourages measurements of fume concentrations in workrooms, without which the proper value cannot be obtained. In absence of a tentative MAC value such tests all too often are not made.

However, while I believe in the use of tentative MAC's, I believe they should be so labelled, so that there will be no excuse for their improper use.

IN SUMMARY then, MAC's are not, in general, too greatly emphasized at the expense of engineering and medical measures. The use of tentative values, based on extrapolation from animal experiments, or even from purely chemical relationships, in absence of other data, is justified. To abandon or sharply curtail this extremely valuable tool of industrial hygiene because it is occasionally misused would be the height of folly.

The Value to the Industrial Physician of Toxicological Information

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IT IS only in relatively recent years that the toxicological laboratory has made its appearance in industries other than in the drug industry and even there, of course, its purpose was one entirely different than the one with which we are concerned today.

The toxicological laboratory in industry is faced with a number of problems. I want to give you a rough idea of the breadth of information required by these problems. dustrial physician, the industrial hygienist,

Problems Presented

FIRST, we consider it an obligation to furnish toxicological information to the in-

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the safety engineer and the designing engineer for the purpose of protecting industrial health within our plant and within the plants of industries which may use our products.

Secondly, we have the obligation to obtain information for the purpose of education on hazards and safe handling of products. This educational program is, of course, directed at immediate plant supervision, foremen and workmen, not only for our own plant but for our customers. The major emphasis of this educational program, however, is directed at the user which includes the general public. The mechanism of dissemination of such information is through precautionary labels which are

placed upon the product package and precautionary information which is furnished as leaflets or handbooks on the operational procedures using such products.

The third class of information is designed for protection during transportation. We must have sufficient knowledge to classify materials according to the Interstate Commerce Commission's classifications so that they will be shipped in the proper containers and have the proper warning labels which are designed specifically for the handler during shipment.

The fourth class of information required of us is somewhat entangled with all others but is specifically information necessary to the legal division of an industry by which it can see that the various and sundry requirements of federal, state and municipal regulations may be faithfully fulfilled.

The fifth category of information which comes from our toxicological laboratories is possibly the one in most demand and requires a great share of our time. It defies simple definition but includes that information which is fundamental to the proper design of a good product. The hazard or lack of hazard of a particular substance within the limitations of the use under consideration is a very important part of the design of the final product.

Toxicity vs. Hazard

IN ORDER to be sure that we will be thinking on a common basis, I would like to define two terms which will be very important to our discussion—these are *toxicity* and *hazard*. Unfortunately these two terms are often confused even by individuals with a great deal of experience in the field. Toxicity and hazard are distinctly not the same. The greatest error is an attempt to delineate hazard directly in terms of a quantitative measure of toxicity.

TOXICITY is the capacity of a substance for causing injury. HAZARD is the probability of that injury occurring in the use of that substance. Our quantitative measure of toxicity is the amount of material required to produce a certain toxic effect when administered in a specified manner.

It will be realized from the definition of our measure of toxicity that there are a number of different figures used to define toxicity. These figures are required because the term *amount* is indicated in several units; grams per kilo of body weight for an oral dose, parts per million by volume in air per time of exposure for air dispersion, per cent in the diet or any one of several other measurements of amount which are required to define practical circumstances of contact. It is also obvious that there are several ways in which the material may be administered depending upon the practical possibility of contact. Each different mode of administration again multiplies the specifications for defining the quantitative aspect of toxicity. As an end result, we have a number of figures, each in a different unit limited by the mode of contact, all of which together are needed to give us a concept of the quantitative aspects of the toxicity of a substance.

This information is of great value in determining the hazard of the substance but it is distinctly not a measure or a statement in itself of the hazard.

The hazard, according to our definition, the probability of injury in use, is dependent not only upon the capacity of a substance for causing injury but to a greater degree on the properties of the substance and the conditions of use which will delineate the probability that the substance in question will be contacted, absorbed and transported to the point within the living organism where its capacity for causing injury may actually result in injury.

Such a philosophy although beautiful to contemplate requires the verification of hard facts. We shall therefore turn here to practical illustrations, the parallel of the case history so dear to the heart of the clinician.

Chloropicrin is a substance well known in the literature as highly toxic and highly irritating; however, it has a relatively low vapor pressure, and the disagreeable and intensely irritating character of its vapor is such as to prohibit voluntary exposure. In actual practice, few serious injuries have occurred. Everyone who has handled it is thoroughly convinced of its disagreeable nature and avoids exposure. The practical hazard then, while far from nil, is low in proportion to its high toxicity.

Somewhat in contrast to this is the commonly known substance, carbon monoxide. Quantitatively this substance is distinctly

less toxic than chloropicrin. It is normally a gas and will diffuse rapidly in the air so that high concentrations are readily attained and it is essentially without warning odor. It is obvious then that although it is less toxic than chloropicrin, it represents a much more serious hazard to the user.

Let us turn to a very different type of problem. Phenol is a very common substance whose toxicity is actually much lower than is usually thought. The hazard in handling this substance, however, is much greater than many substances of much higher toxicity. This fact is due to the great rapidity with which phenol is absorbed through the skin. High concentrations in the blood and central nervous system are attained in a matter of a very few minutes after contact with the surface of the skin. This speed of attainment of a high concentration in the central nervous system may result in rapid death. When phenol comes in contact with the skin or clothing, it is imperative that the clothing be removed and that the individual be placed under a shower or other source of large volumes of water so that the material may be removed at once. By at once, I mean within the first five minutes! Actually when we are fully aware of this problem and the workmen understand it well enough to act promptly, phenol presents very little difficulty. But, for individuals who are not acquainted with this property, phenol may represent a serious hazard much greater than its quantitative toxicity would indicate.

Type of Information Available

Now let us consider some of the types of useful information which the toxicological laboratory should be able to furnish you.

1. The toxicological laboratory can determine quantitatively what level of exposure may result in injury and the type of contact which is of importance; that is, contact with the skin, breathing vapor or dust, or simply by oral ingestion. With this information, the engineer can design a plant to reduce the probability of contact to the point where the hazard is very small. The industrial hygienist and safety engineer, with this information, can verify the plant safety and educate workmen and foremen in the safe handling procedures.

2. The toxicological laboratory can give the industrial physician a great deal of useful information on the type of response to be expected from exposure. You will know where and what to look for following suspected exposure.

3. In many instances the toxicological laboratory can give information on the chemical changes in the body. If there are methods of blood or urine analysis which give a measure of exposure, the toxicological laboratory information will allow the clinical laboratory to check on possible exposure by blood or urine analysis.

Let us take as an example four well-known aromatic hydrocarbons, benzene, styrene, toluene and ethyl benzene.

The greatest hazard from benzene is, of course, the fire hazard. Just because we are more concerned with physiological effects of a substance should not blind us to the fact that the greatest hazard by all means is the hazard of fire. The seriousness of the fire hazard is sharply reduced by the increased alkyl substitution on the benzene ring. The higher alkyl substitutions are still combustible but due to higher flash points, they present a much less significant fire hazard.

Much of the information on the toxicity of benzene was discovered the hard way. It was necessary to kill off a few people and poison a good many more before the necessity for scientific investigation into its physiological effects was realized. I do not need to discuss benzene in great detail as both qualitative and quantitative information in regard to its physiological effects has been available in literature for a long time. It requires very high concentrations of benzene to produce immediate response. The greater problem is physiological injury due to long, continued and repeated exposure to relatively low concentrations. The end results of this chronic exposure is characterized particularly by damage to the hematopoietic system, usually characterized as aplastic anemia.

The chemical changes of benzene within the animal body have been well defined. The compound is oxidized in the body to phenols, conjugated with sulphate and excreted as the ethereal sulphate. It is possible to measure the excretion of the ethereal sulphate, as an index of exposure.

If we now turn to styrene which is a newer compound in commerce, we see that it differs from benzene by the addition of an unsaturated alkyl group. At the beginning of the last war, this compound became of very great significance as a constituent of synthetic rubber. Fortunately, in our company we had the "know how" for large scale production. It is also fortunate that recognizing the potentiality of large scale production of this material, we had toxicological investigations on animals in the laboratory well under way.

Interest in potential hazards of this material was aroused by this increase in production. It is interesting to note that many individuals concluded by analogy that the response would be quite comparable to that observed from benzene and there were a number of medical people determining the ethereal sulphate excretion as a measure of styrene exposure. I will not go into details of the information on styrene but merely give the basic conclusions.

In the first instance, we found that styrene was decidedly less toxic quantatively than benzene. As our long period exposure investigations developed, we realized that styrene did not have any of the physiological effects upon the hematopoietic system observed with benzene. We also found out that styrene went through an entirely different metabolic channel and was not excreted to an appreciable extent as the ethereal sulphate.

In a nut shell, styrene is oxidized in a large degree to benzoic acid and conjugated with glycine and excreted as hippuric acid. We feel that this, to a great degree, accounts for its difference in action and lesser toxicity. In contrast to benzene, styrene has a pungent odor and is sufficiently disagreeable and irritating in concentrations below those which are systemically toxic. It has very good warning properties and we believe no one would voluntarily be exposed to acutely toxic concentrations. At concentrations where we observe physiological response, the response is one of lung irritation and at the threshold of a lethal exposure, the death is usually due to pneumonia following this irritation.

If we turn now to toluene which is a substance old in commerce, we find it is again an alkyl benzene. In the older clinical literature we find a good many reports of blood changes comparable to those following benzene exposure. We find reports of excretion of appreciable amounts of ethereal sulphate. In light, however, of more recent information, we are inclined to believe that the error in these early observations was due to the fact that in those days, toluene contained quite appreciable amounts of benzene. We know that essentially all of the toluene is oxidized to benzoic acid and excreted as hippuric acid. We do not believe that toluene causes the effect on the hematopoietic system observed from benzene. The toxicological effect is probably due largely to respiratory irritation.

The physiological effect of ethyl benzene is very comparable to the other alkyl substituted benzenes. It is oxidized to phenylacetic acid conjugated with glycine and excreted as phenylaceturic acid.

There has been some criticism in clinical quarters of information obtained solely from experimental animals. Admitting all the well known problems of differences in the response of different species, the information obtained gives us a great deal on which to work.

You may hear of cases where animal experiments appear to have missed the point. Although great improvements have been made, we still have much to learn about the experimental approach. We need more critical experiments on a greater number of animal species.

THE industrial physician must watch closely any new material handled by men for whom he is responsible. However, he has a better chance of catching a problem more promptly if he has indicative information from the toxicological laboratory. With improvement in the information from toxicological research, it should be possible for the designing engineer, industrial hygienist and safety engineer to avoid the problem of hazardous exposure in the majority of cases.

I look forward to the day when on the basis of the information from the toxicological laboratory we can design and operate our plants and handle our products with such safety that it is not necessary to learn by the unfortunate injury of our workmen.